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PB-288 198

Mercury, Lead, Arsenic, and Cadmium in Biological Tissue. The Need for Adequate Standard Reference Materials

(U.S.) Environmental Monitoring and Support Lab, Las Vegas, NV

Aug 78

United States
Environmental Protection
Agency

Environmental Monitoring
and Support Laboratory
P.O. Box 15027
Las Vegas NV 89114

EPA-600/4-78-051
August 1978

PB 288198

Research and Development

EPA

Environmental Monitoring Series

Mercury, Lead, Arsenic, and Cadmium in Biological Tissue

The Need For Adequate Standard Reference Materials

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TECHNICAL REPORT DATA

(Please read Instructions on the reverse before completing)

1. REPORT NO. EPA-600/4-78-051	2.
3. RECIPIENT'S ACCESSION NO. PB288198	
4. TITLE AND SUBTITLE MERCURY, LEAD, ARSENIC, AND CADMIUM IN BIOLOGICAL TISSUE The Need for Adequate Standard Reference Materials	5. REPORT DATE August 1978
	6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S) Werner F. Beckert	8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS Environmental Monitoring and Support Laboratory Office of Research and Development U.S. Environmental Protection Agency Las Vegas, Nevada 89114	10. PROGRAM ELEMENT NO. 1HD621/1HD621A 11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS U.S. Environmental Protection Agency - Las Vegas, NV Office of Research and Development Environmental Monitoring and Support Laboratory Las Vegas, Nevada 89114	13. TYPE OF REPORT AND PERIOD COVERED Interim Report 14. SPONSORING AGENCY CODE EPA/600/07
15. SUPPLEMENTARY NOTES	

16. ABSTRACT

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The conclusion is reached that there is a need for the preparation of additional biological reference materials encompassing wide concentration ranges of the elements of interest. The parameters of importance for the cost-effective preparation of biological tissue reference materials are discussed. Some plant and animal species are identified which could advantageously be used to prepare this kind of reference material. In an appendix, the concentrations of mercury in plant and animal tissue samples, as presented in the literature, are listed.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Biological accumulation Mercury Lead Arsenic Cadmium Quality assurance Quality control	Standard reference materials Biological reference materials Biological sample analysis Biological tissue analysis Matrix effects Plant tissue samples Animal tissue samples	06A,C,F,H,T 07B,C

18. DISTRIBUTION STATEMENT RELEASE TO PUBLIC	19. SECURITY CLASS (This Report) UNCLASSIFIED	21. NO. OF PAGES 69
	20. SECURITY CLASS (This page) UNCLASSIFIED	22. PRICE PC A04 / MF A01

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

EPA-600/4-78-051
August 1978

MERCURY, LEAD, ARSENIC, AND CADMIUM IN BIOLOGICAL TISSUE

The Need For Adequate Standard Reference Materials

by

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FOREWORD

Protection of the environment requires effective regulatory actions which are based on sound technical and scientific information. This information must include the quantitative description and linking of pollutant sources, transport mechanisms, interactions, and resulting effects on man and his environment. Because of the complexities involved, assessment of specific pollutants in the environment requires a total systems approach which transcends the media of air, water, and land. The Environmental Monitoring and Support Laboratory-Las Vegas contributes to the formation and enhancement of a sound monitoring data base for exposure assessment through programs designed to:

- develop and optimize systems and strategies for monitoring pollutants and their impact on the environment
- demonstrate new monitoring systems and technologies by applying them to fulfill special monitoring needs of the Agency's operating programs

A prerequisite for the generation of reliable analytical data is a sound quality assurance program which in turn depends to a large degree on the skillful use and the availability of appropriate reference materials. This report reviews the present scarcity of standard reference materials consisting of biological tissues and the need for the preparation of additional materials. A cross section of published data is presented demonstrating the wide concentration ranges of mercury, lead, arsenic and cadmium encountered in biological samples. The parameters of importance are identified for the cost-effective preparation of biological reference materials containing elevated levels of toxic elements. This information will be of value to everybody involved in analyzing biological tissues for toxic elements. For further information, the Quality Assurance Branch, Monitoring Systems Research and Development Division, should be contacted.

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ABSTRACT

The present situation of standard reference materials consisting of plant and animal tissues is examined. A brief literature review presents a cross section of published data on the incorporation of mercury, lead, arsenic and cadmium into plant and animal tissues. It points out the wide concentration ranges of these elements that are encountered in biological tissue samples under environmental and experimental conditions. These concentration ranges are compared with the individual values of the corresponding elements as determined for the biological standard reference materials presently available from the National Bureau of Standards.

The conclusion is reached that there is a need for the preparation of additional biological reference materials encompassing wide concentration ranges of the elements of interest. The parameters of importance for the cost-effective preparation of biological tissue reference materials are discussed. Some plant and animal species are identified which could advantageously be used to prepare this kind of reference material. In an appendix, the concentrations of mercury in plant and animal tissue samples, as presented in the literature, are listed.

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ACKNOWLEDGEMENT

Permission by Dr. C. C. Patterson, California Institute of Technology, to include data from an as yet unpublished report is gratefully acknowledged.

INTRODUCTION

In the years ahead, increasing amounts of toxic stable and radioactive pollutants will be introduced into the environment. A major part of this increase will come from the combustion and conversion of fossil fuels, including oil shale, and from relatively new energy sources such as nuclear and geothermal. In addition, waste disposal via incineration will contribute significant amounts of pollutants such as cadmium and zinc.

In order to determine the patterns and the extent of environmental pollution, pollutant levels in a variety of environmental matrices must be monitored. Valid laboratory analyses and associated quality assurance procedures attendant to the monitoring of pollutants depend on the availability and the use of standard reference materials.

Standard reference materials (SRM's) are generally materials which have been certified for one or more (physical or chemical) parameters. Examples are alloys, ores, radioactivity standards, polymers and biological tissues. The major uses of SRM's are as control materials (to be analyzed periodically along with unknown samples), as bases for the calibration of instruments, and as materials for technique and instrument development and evaluation. Additional uses of SRM's are in methods standardization and equivalency determinations, cross-check programs, and laboratory performance evaluations.

There is an important limitation to the use of SRM's, which is particularly true for biological material. It has been found that, for certain constituents, the matrices* of materials analyzed can influence the validity of the analytical results (Zief and Mitchell, 1976). Therefore, to minimize the occurrence of matrix effects, both the SRM's used to produce the calibration curves and the samples to be analyzed must have similar matrices to assure that the instrument responses are similar for the interferences from the matrix (Cali et al., 1975). This is of utmost importance since the analytical data must be scientifically acceptable and legally defensible.

There is presently a scarcity of adequately characterized biological SRM's that contain pollutants of current interest at varying levels. In this report the present SRM situation with respect to biological tissues is discussed. A cross-section of published data demonstrating the wide concentration ranges

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* Matrix in this context means the combination of chemical composition and physical structure of the sample material.

of mercury, lead, arsenic and cadmium encountered in biological tissues is presented, and the parameters of importance for the cost-effective preparation of biological SRM's containing elevated levels of mercury, lead, arsenic, and cadmium are identified.

THE STANDARD REFERENCE MATERIAL SITUATION

The National Bureau of Standards (NBS), the world's leading producer of SRM's, has approximately 900 SRM's presently available (National Bureau of Standards, 1975a, 1975b) with approximately another 100 in preparation. Because of the rapidly increasing demand, the NBS can only partially fulfill the requests for new SRM's (Cali et al., 1975).

The NBS has recognized the need for biological SRM's which are certified for in vivo incorporated elements and has started a program for their production which began several years ago. The biological SRM's presently available from the NBS include the following:

SRM 1569 - Brewers Yeast, certified for chromium;
SRM 1570 - Trace Elements in Spinach, certified for 16 elements;
SRM 1571 - Orchard Leaves, certified for 19 elements;
SRM 1573 - Tomato Leaves, certified for 14 elements;
SRM 1575 - Pine Needles, certified for 15 elements;
SRM 1577 - Bovine Liver, certified for 12 elements.

Biological SRM's presently under preparation or consideration by the NBS include wheat flour, rice flour, and oyster meal. All of the SRM's listed above contain environmental levels of the certified elements (National Bureau of Standards, 1975b). Table 1 reports the certified values of these SRM's for mercury, lead, arsenic, and cadmium. The preparation of the plant tissue SRM's involved handpicking the plant material, removing the stems and other undesirable parts, and freeze-drying, grinding, sieving, blending, sterilizing (with radiation), and analyzing the material (Taylor, 1976).

The preparation and certification of SRM's are time-consuming and expensive (Cali et al., 1975), because SRM's must satisfy a number of important requirements. Solid SRM's to be used for chemical analyses must be of a small particle size because there is usually no guarantee that the starting material is microuniform. A high degree of uniformity is required to keep the minimum reproducible sample size as small as possible (the NBS usually specifies the minimum sample size which will still guarantee reproducible results). Certified SRM's must be stable for extended periods of time under the proper storage conditions. The material must be analyzed and certified by at least two independent methods. In order to exclude errors caused by matrix effects, the matrix of a SRM should ideally be similar to the matrix of the sample to be analyzed with the constituents of interest incorporated in an identical manner in both the SRM and the sample. Furthermore, because

TABLE 1. CERTIFIED VALUES FOR MERCURY, LEAD, ARSENIC AND CADMIUM IN SRM's OFFERED BY THE NBS (μg/g)[1]

	Hg	Pb	As	Cd
Orchard Leaves	0.155	45[2]	14[2]	0.11
Tomato Leaves	(0.1)	6.3	0.27	(3)[3]
Spinach	0.030	1.2	0.15	(1.5)[3]
Pine Needles	0.15	10.8	0.21	(<0.5)[3]
Bovine Liver	0.016	0.34	(0.055)	0.27

[1]Values in parentheses are not certified but are given by the NBS for information only.

[2]The lead and arsenic values in the orchard leaves are relatively high because of past applications of lead- and arsenic-containing pesticides to the orchard.

[3]Cadmium was not sufficiently homogeneous for certification.

one-point calibration may be unreliable and extrapolation may result in erroneous conclusions, calibration curves should be prepared using two or more SRM's containing different levels of the element or elements in question to closely bracket the unknown value for interpolation from the calibration curve.

With increasing emphasis on the use of biological monitors, an increasing number of environmental samples will consist of various plant or animal tissues. This, in turn, will require the availability of a larger variety of biological SRM's so that the influence of matrix effects on analytical data can be minimized.

To meet the requirement for varying quantities of given pollutants in the reference material, it is practical to select a single or a few plant and animal species into which the desired pollutants can be systemically incorporated. In order to minimize the cost per sample unit, it is further desirable to prepare and certify as large a batch of material at one time as is feasible. In addition, it would be very expensive to prepare a series of biological SRM's for each pollutant which differ only in concentration. Therefore, the exploration of alternate approaches is justified.

A program has been initiated at EMSL-LV to prepare biological SRM's containing a variety of in vivo incorporated pollutants. These SRM's will

be useful in the analysis of biological samples obtained by monitoring activities in the vicinity of fossil fuel extraction and utilization plants, geothermal sites, etc. The first set of pollutants to be incorporated under this program into plant and animal tissue consists of mercury, lead, arsenic, and cadmium which are of particular interest and ever-growing concern. The measurement methodology for these elements is fairly well established, but there is presently only a small number of biological reference materials available. Other pollutants of present and future concern to the Agency will be incorporated into plant and animal tissue at a later date and processed to form SRM's.

MERCURY, LEAD, ARSENIC, AND CADMIUM IN PLANT TISSUE

The chemical analysis of plant tissue usually does not indicate whether the elements found have been taken up by the plant and have become incorporated into the plant tissue, or whether they have merely been deposited on the plant surface as a result of air pollution (Shaklette, 1970). Yet, this distinction is of interest because materials that have been deposited on aerial plant parts often consist of oxides or other relatively insoluble chemical compounds. But elements that have been incorporated into plant tissue allegedly exhibit a higher biological availability. It is also probable that the volatility of incorporated substances is in many instances different from that of surface-deposited substances. This is an important consideration which will directly affect the stability and shelflife of SRM's.

Many different species of plants have been investigated to determine the uptake and incorporation of mercury, lead, arsenic, cadmium and many other pollutants into their tissue, as evidenced by numerous original and review articles. It would be redundant to list and discuss all of them; however, examples of data on the uptake of mercury by plants are listed in Appendix A, Tables 1 through 8. These data have been obtained from analyses of plant tissues grown under a variety of environmental and experimental conditions, and their ranges should be indicative of the mercury levels which might be expected for environmental samples collected from the clean to the highly polluted areas. Additional uptake data for mercury as well as lead, arsenic, and cadmium will be discussed in the following pages.

MERCURY

Mercury enters the environment through natural weathering processes and through a number of man-related activities. It has been said that natural weathering processes and man-caused processes contribute approximately equally to the global mercury contamination (Klein, 1972). However, mercury entering the environment via natural processes usually results in low concentrations over wide areas whereas mercury as a contaminant enters the environment in few locations but at high discharge rates and concentrations. Mercury uses which result in significant losses to the environment are in the chloralkali industry, in the electrical apparatus and industrial control instrument industries, in general laboratories using mercury, in the paint industry and, at least in the past, in mercury-containing pesticides (D'Itri, 1972a). Some of these uses and the resulting losses to the environment have been sharply curtailed during the last few years. Other important environmental mercury sources are the combustion of fossil fuels and smelter emissions and effluents.

Discharges of volatile mercury into the environment can be deposited on aerial plant parts or in soil and can eventually enter plants via the root system. Translocation of mercury occurs within plants. Lindberg (1961) has shown that foliar application of phenylmercuric acetate to rice plants resulted in the translocation of mercury to the grain, and Smart (1968) showed that mercury was translocated to fruits, tubers, and seeds after foliar application of mercury fungicides. D'Itri (1972b), in his review summarizing data on mercury translocation in plants, reported that translocation occurred for apples, potatoes, tomatoes, grains, and other crop plants.

Foliar absorption can be an important entryway for mercury into plants even when the mercury compounds have been applied only to the soil or, in the case of aquatic plants and hydroponic cultures, to the water. A variety of inorganic and organic mercury compounds can be reduced in an aquatic or terrestrial environment to the metallic or even the methylated state (Tonomura et al., 1968; Wood et al., 1968; Beckert et al., 1974). These volatile forms of mercury can easily evaporate from the soil and water surfaces and at least be partially deposited on nearby plant surfaces, thus becoming available for foliar absorption. Obviously, because of these reduction and methylation processes, the roots of edaphic and aquatic plants are exposed not only to the chemical form of mercury as applied to the soil or water but also to methylmercury and elemental mercury, and possibly other chemical forms.

LEAD

Lead is of high environmental priority because it is being continuously introduced into the environment in large amounts. The most important source of environmental lead pollution is automotive exhaust which comprises the combustion products of automobile fuels containing lead compounds for pre-ignition prevention (according to Hall (1972), approximately 20 percent of the U.S. total consumption of lead in 1969 was used for gasoline additive production). During the combustion process, most of the organic lead is converted to inorganic compounds and is emitted as hydroxide, halide, and oxide, together with smaller amounts of carbonate and sulfate (Habibi, 1970; Ter Haar and Bayard, 1971). An important source of lead (as well as arsenic) to the environment was the application of lead arsenate as an insecticide. This use has now been largely discontinued. However, in certain agricultural areas, large amounts of lead arsenate have been added to the soil over several decades, and both accumulated lead and arsenic will present a problem for some time. Further lead sources of environmental concern are inorganic fertilizers containing lead as impurities. Lead deposition from fertilizers may even account for the high lead levels in certain agricultural soils (Schuck and Locke, 1970) which have been reported to range up to 200 ppm (Swaine and Mitchell, 1960). Smelter emissions and sewage sludge, as well as lead-based paint pigments and storage batteries, also contribute to lead pollution.

Plants can absorb and translocate lead. This fact has been considered for lead prospecting (Cannon 1960, 1971). Root uptake is an important pathway of lead uptake in plants in the field as has been demonstrated by a number of

researchers (for additional references see Zimdahl and Arvik, 1973). Ter Haar et al. (1969), for example, found that 46 percent of the lead content of perennial rye grass blades and all of the lead found in radish roots came from the soil rather than from the air. Lead levels in the edible parts of crop plants are usually well below 1 ppm (Schuck and Locke, 1970) and are lower than those of the other vegetative plant parts (Aarkrog and Lippert, 1971). For a variety of plants, an average lead content of 10 ppm (dry weight) was reported with a high of 45 ppm in potato tops (Warren and Delavault, 1962). Gamble (1963) reported that the leaves of various plant species in wooded areas contained from less than 0.3 ppm to as much as 30 ppm (dry weight) of lead, and Prince (1957) found 10-25 ppm (dry weight) in corn leaves.

Aerial contamination, however, occurs to some extent in almost all cases and becomes the dominating factor especially in areas close to heavily traveled highways or to smelters (Zimdahl and Arvik, 1973). Hay contains normally 2 to 3 ppm (dry weight) of lead, but in areas near smelters values up to 284 ppm (dry weight) have been determined (Hammond and Aronson, 1964). In one study it was shown that 40-50 percent of the total lead associated with plants could be removed by one washing with distilled water, whereas two washings removed between 60 and 70 percent (Page et al., 1971).

Only three years ago it was believed that the methylation of inorganic and organic forms of lead could not occur in the environment (Wood, 1974). Recently, however, methylation of lead has been shown to occur but under conditions which are unlikely to make it a problem similar to the methylation of mercury (Wong et al., 1975).

ARSENIC

Arsenic is suspected of being carcinogenic to humans and being related to arteriosclerosis and chronic liver diseases (Hueper, 1963; Wagner, 1973). There still remains some doubt as to these allegations, but the potential hazard to man makes it necessary to monitor the distribution and the fate of arsenic in the environment.

Arsenic is ubiquitous in nature, with concentrations in uncontaminated soils ranging from 0.2 to 40 ppm (Olson et al., 1940) with an average of about 5 to 6 ppm. It is concentrated in a variety of minerals including many sulfides and phosphates; coal reportedly can contain up to 2000 ppm (Onishi, 1969; Bowen, 1966; Boyle and Jonasson, 1973). Large amounts of arsenic can be distributed over areas adjacent to facilities engaged in processing arsenic-containing ores so that arsenic levels can be reached that are directly injurious to humans (Oyanguren and Perez, 1966; Birmingham et al., 1965). Inorganic and organic arsenicals are still being used as pesticides; extensive applications in the past gave rise to very high arsenic levels in some soils, especially those in orchards. The burning of coal constitutes another major source of arsenic. It was reported that fly ash from a coal-burning power plant contained as much as 139 ppm of arsenic (Furr et al., 1976a).

Arsenic emission to the environment was reduced considerably during recent years, mainly by abatement procedures applied to industrial sources of arsenic. Probably the most important factor was the replacement of arsenical pesticides, especially sodium arsenite, which was banned (Fed. Reg., 1968) and lead arsenate which was largely replaced by organic pesticides.

Arsenic compounds can be methylated leading to a number of very toxic compounds (Wood, 1974), but it is unlikely that these transformations will pose a serious problem in the environment.

The literature on arsenic uptake by plants is much less voluminous than that on mercury or lead. Plants vary in their tolerance, and in a few instances, very high levels of arsenic have been reported for certain plants. Warren et al. (1968) reported 10,000 ppm in the ash of Douglas Fir needles which would correspond to a value of nearly 1,000 ppm on a dry weight basis. Even higher values were reported for native vegetation collected from arsenic-enriched sites (mine wastes), namely 6,640 ppm (dry weight) for *Jasione montana L.* and 4,130 ppm (dry weight) for *Calluna vulgaris (L.)* Hull (Porter and Peterson, 1975). Fortunately, however, the edible parts of plants usually do not accumulate hazardous levels of arsenic and contain considerably less than the permissible limit of 2.6 ppm (U.S. Dept. of Agric., Pestic. Reg. Div., 1968). Furr et al. (1976a) found 0.2 ppm of arsenic (dry weight) in the edible parts of beans, cabbage and carrots, and lower values in a number of other vegetables when grown on fly-ash-amended soil with an average arsenic content of 16 ppm. Steevens et al. (1972) grew potatoes in fields where the potato vine defoliant sodium arsenite had been used extensively in the past. The soil contained 2.7 to 25.7 ppm of arsenic while from the harvested potatoes the tuber peeling contained 0.2 to 2.6 ppm (dry weight) and the tuber flesh only up to 0.6 ppm (dry weight). Chisholm (1972) conducted similar experiments with vegetables grown in soil which had been heavily treated with lead arsenate and contained, at the time of the experiment, 122.5 ppm of arsenic. None of the arsenic levels which he reported were above 1.1 ppm (dry weight) in the edible parts of the plants. This is in line with older observations by McLean et al. (1944) who reported that vegetables grown on soils treated with high levels of lead arsenate seldom contained more than 1 ppm of arsenic in the edible parts.

The surface of aerial portions of plants may be contaminated with resuspended material when soils nearby had been treated with substantial quantities of arsenic. Jones and Hatch (1945) found 3.1 ppm of arsenic in the aerial growth of vegetable plants which were growing in untreated soil adjacent to arsenic-treated fields whereas the roots of the plants from the untreated soil contained only 1.1 ppm of arsenic. Obviously, the aerial portions must have been contaminated by arsenic-containing dust particles since roots usually accumulate more arsenic than the aerial plant parts. Some of the data in the literature on plant uptake of arsenic might therefore be questionable unless the plant tissues were washed carefully prior to analysis to remove adsorbed soil particles.

CADMIUM

Cadmium is of concern as an environmental pollutant mainly because of its relatively high toxicity to animals and humans. The kidneys are the critical organ with respect to prolonged low-level exposure to cadmium (Friberg et al., 1973). An association seems to exist between cadmium exposure and the incidence of cancer in man (Friberg et al., 1971). In animal experiments, cadmium salt injections resulted in malignant tumors. Cadmium has also been shown to be teratogenic in animals (Friberg et al., 1973; Mulvihill et al., 1970; Chernoff, 1973), as well as mutagenic (Doyle et al., 1974; Shiraishi and Yosida, 1972); however, the mechanism is unknown.

Cadmium is closely related chemically to zinc and is always found in zinc ores. While zinc is an essential trace element in living cells, cadmium is very toxic. Cadmium is also contained as an impurity in phosphate rock with concentrations ranging as high as 100 ppm (Williams and David, 1976), in coal from 0.02 to 10 ppm (Hiatt and Huff, 1975) with values as high as 28 ppm (Gluskoter and Lindahl, 1973), and from 0.42 to 0.53 ppm in heating oil (Hiatt and Huff, 1975).

Most of the commercially produced cadmium is recovered as a by-product during the refining of zinc and other metals. The principal uses are in electroplating, which accounts for approximately 50 percent of the total cadmium production, as a component of stabilizers for plastics, in pigments and alloys, and in cadmium batteries.

The recycling of cadmium is difficult, if not impossible, for most of the cadmium-containing products; therefore, the amount of cadmium released to the environment from the disposal of these products is an important consideration. Other factors in environmental cadmium pollution are the use of phosphate fertilizers, the combustion of fossil fuels, and the processing of many metal ores, especially those of zinc, lead, and copper which contain small amounts of cadmium. A source of growing concern is sewage sludge which reportedly has cadmium levels as high as 200 ppm (Furr et al., 1976b), and geothermal sites, a largely unknown factor in environmental pollution.

Airborne and waterborne cadmium as well as fertilizer and sewage sludge can substantially increase cadmium concentrations in soil. Plants take up cadmium via roots and foliage, but little is known about the uptake mechanism. In experiments with culture solutions containing cadmium chloride, it was found that the cadmium concentrations were always greater in the roots than in the shoots. It was concluded that the roots can take up large quantities of cadmium from culture solutions, but that there are apparently mechanisms which restrict the movement of cadmium through plants (Jarvis et al., 1976). As much as 14.95 µg/g of cadmium were reported for corn plants (excluding roots) which were grown on soil amended with cadmium sulfate to a level of 5.0 µgCd/g soil (Street et al., 1977). Similar accumulations were reported by John et al. (1972), who studied cadmium uptake by radish and lettuce plants grown on a variety of soils amended with cadmium chloride to a level of 100 ppm. They found that the edible portions of the radishes and lettuce harvested from 30 different soils averaged 387 ppm and 138 ppm (dry weight),

respectively, and concluded that potentially hazardous accumulations of cadmium in plant tissues may occur when soils are contaminated.

Williams and David (1976) reported that fertilizer impurities had increased the original levels of hydrochloric acid-soluble cadmium by more than an order of magnitude in several soils and that this resulted in most cases in considerable increases in the cadmium content of plants grown on such soil. Furr et al. (1976a) in their study on the effects of fly-ash amendments to soils on the uptake of certain elements by vegetable plants reported that the cadmium content of the plant tissues did not increase. The plant values of cadmium on a dry weight basis from the control and amended soils containing 0.1 ppm and 0.14 ppm of cadmium, respectively, were as follows (reported in ppm): beans 0.1/0.1, cabbage 0.2/0.2, carrots 1.1/0.6, millet 0.2/0.2, onions 0.6/0.4, potatoes 0.4/0.2 and tomatoes 0.1/0.1. The results are not surprising when one considers that there was only a slightly higher cadmium level in the amended soil. These authors reported in another study (Furr et al., 1976c) that the same vegetable plants took up high amounts of cadmium from soil amended with 10 percent of municipal sludge containing 112 ppm cadmium. For the control soil with <0.9 ppm and the amended soil with 11.3 ppm cadmium, the plant values of cadmium on a dry weight basis were as follows (in ppm): beans 0.1/1.8, cabbage 0.2/37.5, carrots 1.1/3.9, millet 0.2/24.5, onions 0.6/9.2, potatoes 0.3/2.0, and tomatoes 0.1/2.4.

Mushrooms can accumulate relatively high amounts of cadmium from soil. Stijve and Besson (1976) reported that *Agaricus edulis* grown on soil containing 0.16 ppm cadmium contained 2.1-7.5 ppm cadmium (dry weight) which corresponds to a concentration factor of 13 to 47. Other mushrooms of the same genus which were collected from various areas in Europe and analyzed by these investigators contained from 0.1 to 75 ppm cadmium.

Aerial contamination was shown to occur by Little (1973) who analyzed elm leaves collected in an area with heavy cadmium fallout. He reported that more than 60 percent of the total cadmium in the elm leaves could be removed by washing with deionized water, and more than 98 percent with 1 percent HNO_3.

Methylation of cadmium will not be a problem as cadmium alkyls hydrolyze under environmental conditions.

CONSIDERATIONS FOR THE PREPARATION OF PLANT TISSUE SRM's

The concentrations of toxic elements in plant material which will be encountered by the analyst comprise a wide range, from low levels in samples from certain pristine areas to very high levels in plants grown in highly contaminated or experimentally enriched soils. Figure 1 shows graphically the concentration ranges of mercury, lead, arsenic and cadmium in plant tissues, as discussed in this report. Included are, for comparison, the concentrations of the same elements in the plant tissue SRM's available from the NBS.

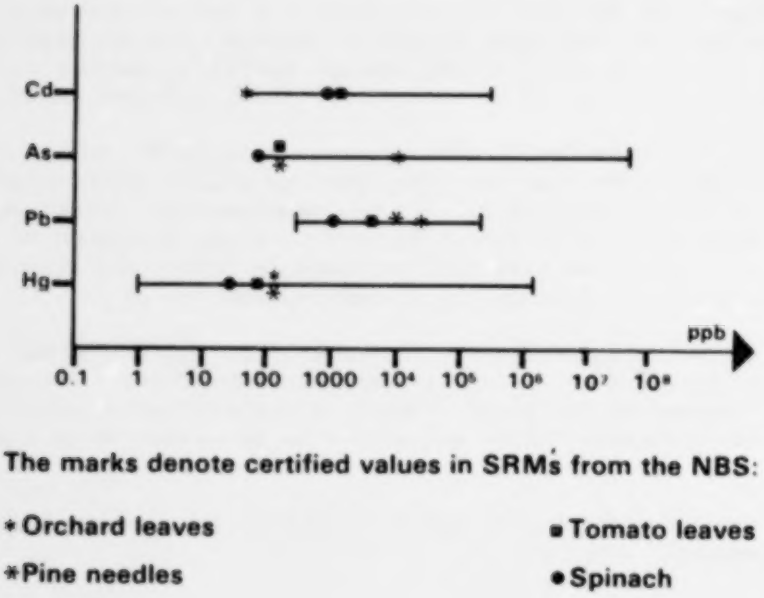

Figure 1. Ranges of mercury, lead, arsenic, and cadmium levels as reported in the literature for plant tissues.

It is apparent that especially for mercury and arsenic, concentration ranges extending over several orders of magnitude are not covered by SRM's. In order to fill these gaps, a series of SRM's with varying pollutant concentrations must be prepared. It seems that the most cost-effective way of achieving this would be to prepare batches of highly contaminated and uncontaminated (except for background levels) plant tissue of the same kind and, after separate processing, blend them to the desired pollutant concentrations.

A plant species suitable for cost-effective in vivo incorporation of pollutants and processing to form a SRM should fulfill certain requirements. It should tolerate and incorporate as many of the projected pollutants as possible because the experience gained during early studies may directly apply to the incorporation of additional pollutants into the same kind of plant. The plant should grow fast because large quantities of fresh plant tissue will be needed to prepare enough material to justify the certification cost. Harvesting should preferably be a continuous operation because of the logistics involved in picking and processing of the plant material. In order to obtain a homogeneous SRM, a uniform starting material is necessary. Large leaves with few veins would therefore make a suitable starting material since only the stems and large veins will have to be removed. It must be possible to easily monitor and eventually modify the pollutant levels in the growth medium; this makes the use of hydroponic culture techniques almost imperative. Obviously, a plant species is required which grows well and is easy to handle in large quantities under these production conditions.

The above criteria eliminate the vast majority of plants from consideration as suitable matrices for SRM's containing in vivo incorporated pollutants. Algae and other submerged plants or plant parts may adsorb rather than incorporate the pollutants. Certain fungi accumulate heavy metals and other pollutants, but their growing conditions are uncertain and are, in general, not sufficiently defined. Mosses are slow growers, and it would be difficult to avoid cross-contamination from spiked growth media. Tree leaves have been used as a SRM, but picking and processing were found to be extremely labor-intensive processes. Fruit, grain, and plant parts such as tubers and roots are not representative of the plant tissue likely to be encountered in future monitoring activities. Some of the plants often used in research, such as alfalfa, peas, and barley, are impractical since extensive manual labor is necessary to isolate the usable leaf parts.

Plants which meet many of the above requirements are leafy vegetables such as cabbage, lettuce and spinach. These can be grown under controlled conditions with the planting staggered for continuous harvesting. Even more promising are leafy water plants such as water hyacinths that are self-propagating, require very little care, and produce leaves which are relatively easy to process since only the major veins must be removed.

The water hyacinth (*Eichhornia crassipes*) is basically an undesirable aquatic weed which thrives in water bodies of tropical and subtropical regions. Its growth aspects have been well documented, and the interest in water hyacinths is evidenced by the number of articles published in the Hyacinth Control Journal, a research publication which is devoted exclusively to aquatic weed research. The prolific growth of water hyacinths in water bodies is only limited by the availability of nitrogen and phosphorus (Wahlquist, 1972). Their growth rate under optimum conditions is high. Boyd (1976) reported from a series of experiments an average growth rate of 194 kg/ha/day over a 5-month period; however, values as high as 540 kg/ha/day have been reported for growth in a eutrophic lake (Yount and Crossman, 1970).

A number of uses for water hyacinths have been recently considered. To reduce the nutrient levels of water bodies the cultivation of water hyacinths followed by their periodic removal has been proposed for eutrophic lakes which have high concentrations of nitrogen and phosphorus (Boyd, 1976). The potential of water hyacinths for the removal of pollutants from sewage was investigated by Wolverton and McDonald (1975a), and Wooten and Dodd (1976). The recovered plant material could possibly be used as animal feed (Hentges et al., 1972; Wooten and Dodd, 1976) or it could be used for the production of methane (Wolverton et al., 1975).

The growth of water hyacinths under laboratory and field conditions in the presence of a variety of pollutants has been studied by Wolverton and his group (for references see Wolverton and McDonald, 1975a). They found that mercury, lead, cadmium and several other metals, as well as phenols and certain pesticides, were efficiently removed from solution. Approximately 10 percent of the removed metals were transported to the aerial parts of the plants (Wolverton and McDonald, 1976).

Water hyacinths are easy to grow in hydroponic solution. Their large leaves, which are above the water surface, could be harvested periodically and processed to form a uniform powder that is suitable as a SRM. These and other advantages mentioned earlier make water hyacinths grown under hydroponic culture conditions a very desirable plant species for the in vivo incorporation of toxic elements into plant tissues. Time and additional studies will show if one kind of plant tissue SRM which contains pollutant levels above ambient will be sufficient for future analytical tasks, or if a variety of plant tissue SRM's will have to be developed.

MERCURY, LEAD, ARSENIC, AND CADMIUM IN ANIMAL TISSUE

Standard reference materials consisting of animal tissues containing in vivo incorporated toxicants are needed for a variety of tasks. It was pointed out earlier that the use of biological receptors as investigative tools is increasing in environmental pollution monitoring studies. As a consequence, an increased number of tissue samples from indigenous and domestic animals must be collected and analyzed for a variety of pollutants. In addition, tissue samples from domestic animals raised in highly polluted areas, such as the vicinity of smelters or heavily traveled highways, which may have excessive amounts of toxic elements incorporated into their tissues will have to be analyzed.

A variety of different organ tissues is desirable as SRM's since in many routine cases only one kind of tissue might be obtained, because it is either easily accessible or is part of a target organ for a particular pollutant. Examples of target organs or tissues which are frequently sampled for analysis are liver, kidneys, blood, and muscle. Some of these tissues, such as blood, might even be routinely used to screen humans. A special case is hair, a metabolic end product that can be painlessly collected and easily handled, stored, and analyzed. Hair can be considered as a minor organ for the elimination of certain elements. Although its use in monitoring exposure to and accumulation of toxic elements is still controversial, it might become feasible in special problem areas.

Animals take up varying amounts of toxic elements with their food and with air, as well as by licking and grooming, soil ingestion, and other activities. Usually only a relatively small percentage of the ingested toxic elements is incorporated into the animal body organs while the remainder is excreted. The amount retained depends on the animal, its age and health, the feed, the chemical forms of the toxic elements, and other factors. Different tissues accumulate different levels of the toxicants; these levels are dependent on the nature of the toxic elements and, in some cases, on their chemical form and the mode of entry.

MERCURY

Essentially all animal tissues contain low levels of mercury. A large number of epidemiological studies have been carried out, and vast amounts of data have been published on the mercury content of various animal tissues. It was found that, in general, animals higher up in the food chain accumulate comparatively higher amounts of mercury. Tables 9 through 12 list a cross section of data on mercury levels reported for fish, birds, and mammals from

a variety of studies; these values illustrate the range which might be encountered in future experimental and monitoring studies.

The rates of absorption, distribution, excretion, and toxicity of mercury in animals can be influenced drastically by the chemical form of the applied mercury. Experiments with mice showed that only about one percent of ingested inorganic mercuric compounds but up to 98 percent of methylmercury were absorbed via gastrointestinal tract (Hartung and Dinman, 1972). It has been discussed that inorganic mercury might be converted to organic mercury in man by the intestinal flora or by biochemical processes, but no methylmercury has been detected thus far in mammals following ingestion of inorganic mercury (Hartung and Dinman, 1972). Organomercurials are generally more likely than inorganic mercurials to cause genetic, teratogenic, and carcinogenic effects. Phenyl- and methylmercury are 200 times more effective as c-mitotic agents than inorganic mercurials and 1000 times more effective than colchicine, the classic inducer of c-mitosis (D'Itri, 1972a). Treatment of human leucocytes in culture with 1 to 2.0×10^{-6} M methylmercuric chloride solution resulted in c-mitosis (D'Itri 1972b). Results of animal experiments indicated that organic mercury compounds directly affect either the genetic material, causing chromosome breakage (Ramel, 1969), or meiosis (Ramel, 1967; Ramel and Magnusson, 1969), and even produce mutagenic (Ramel, 1969) or teratogenic effects (WHO, 1966) or sarcomas (Druckery et al., 1957).

The mercury content of fish has been of major concern since Canada announced in March of 1974 that 12,000 lbs of commercially caught walleye from Lake St. Clair were to be destroyed because of mercury contamination. This triggered a chain reaction of fishing closures and restrictions in North America and marked the beginning of the "mercury scare" for the United States.

The mercury in fish muscle is present primarily as methylmercury, thus making it a serious hazard to man. The methylmercury levels depend, as expected, on the mercury concentration of the water. Predatory fish such as tuna and swordfish have generally higher levels. For mercuric chloride in water, the lethal concentrations for fish range from 0.02 ppm for guppy to 9.2 ppm for rainbow trout (U.S. Environmental Protection Agency, 1973). However, even in heavily polluted bodies of water, the mercury concentration is usually not high enough to be lethal to fish. Mercury levels reported for freshwater fish vary from 0.08 ppm for brown trout (Byrne et al., 1971) to 27.8 ppm for northern pike (Fimreite and Reynolds, 1973). For marine fish the values ranged from 0.02 ± 0.01 ppm for sardines (average of 104 FDA analyses, reported by Simpson et al., 1974) to 14.0 ppm for Pacific blue marlin (Rivers et al., 1972). Fish and shellfish from the highly polluted Minamata area contained 9-24 ppm of mercury (Holden, 1973).

The mercury levels found in birds reflect their dietary habits. As expected, birds eating fish from polluted water, or grain and seeds from agricultural areas where mercury-containing pesticides have been used, contain higher mercury levels than birds living in areas of low mercury concentrations. This is documented in a paper by Spronk and Hartog (1970) who reported that mercury levels in flight feathers of goshawks and buzzards in the Netherlands varied from 26 to 72 ppm and 2 to 23 ppm, respectively. The high

levels in the feathers of the goshawk were related to their diet, one third of which was seed-eating pigeons, whereas buzzards prey mainly on sprout- and root-eating mice. Martin and Nickerson (1973) found that starlings which were collected in the United States from 50 sites had mercury levels below 0.50 ppm with 76 percent of the birds containing 0.05 ppm or less. Kreitzer (1974) sampled mourning doves in the Eastern United States and found that the breast muscles of 93 percent of the birds contained less than 0.05 ppm of mercury. Analyses of different bird organs generally showed mercury concentrations decreasing in the order of liver-kidney-muscle-brain (Westermark, 1967; Stoewsand et al., 1971). Gardiner et al. (1971) found in chickens which were fed ^{203}Hg-methylmercuric dicyandiamide that the radioactive isotope was concentrated in the liver and kidneys.

The mercury levels in mammalian tissues vary widely (Appendix A, Table 12) and reflect to a great extent the dietary habits of the animals. In areas of mercury contamination the levels of mercury increase upward with the food chain which makes predators quite suitable for environmental monitoring purposes. As a food source for man, wild mammals are only of very minor importance in the United States.

The mercury intake by domestic animals can be largely controlled via their feed, and their mercury levels should normally be around background levels. The isolated incidents reported of humans being poisoned through the consumption of mercury-containing meats resulted from accidents or ignorance. Such a tragic case happened in New Mexico and resulted in death or blindness to members of a family that had consumed the meat of hogs which were fed waste seed grain treated with the fungicide methylmercuric dicyandiamide (Curley et al., 1971).

The distribution of mercury in mammalian tissue depends on a number of factors such as route of entry and chemical form of the mercury compound, dietary content of certain other elements like selenium (Ganther et al., 1972), and age of the animal. Experimental studies with mercury-203 showed that the mercury level was higher in liver and kidney than in other tissues for a variety of mammals including cows, calves and goats, and that the level was independent of the chemical form or the route of dose administration (Ansari et al., 1973; Friberg and Vostal, 1972; Potter et al., 1972; Sell and Davison, 1973; Stake et al., 1975). However, the relative distribution among the tissues may vary with the chemical form used and the route of entry. In calf muscle, the ratio of methylmercury to mercuric chloride was 594 when the compounds were administered orally, and only 6 when introduced intravenously (Ansari et al., 1973; Stake et al., 1975).

It has been claimed that hair analysis is a good technique for monitoring mercury levels in the human population (Hartung and Dinman, 1972). Hair has been used in recent studies as an index of mercury exposure for fish consumers in Ontario (Jervis et al., 1970), and Takeuchi (1972) quoted a Japanese publication which reported that a close relationship existed between mercury concentration in hair and the onset of the Minamata disease. Eads and Lambdin (1973) determined mercury and six other elements in selected human hair samples from an area with refineries and petrochemical plants. A wide range

in content for both males and females was observed for mercury, lead, and zinc. The average mercury concentration was found to be 5.4 ppm for males and 5.5 ppm for females.

Before hair samples can be used as a reliable exposure monitor for mercury, several important parameters must be more clearly defined. The reliability of removal of surface contamination, and the establishment of a hair-blood ratio relationship are particularly important.

LEAD

Lead contamination is more widespread than mercury contamination, but fewer spectacular cases of acute poisoning or contamination have been reported. The presence of lead in the environment must nevertheless be monitored primarily for its persistence and its long-term effects. Lead is present in all animals, and as for all heavy elements, animals higher up in the food chain accumulate higher amounts of lead in their bodies. Organic lead compounds are generally more toxic than inorganic ones (but the difference is not as pronounced as in mercury compounds). This higher toxicity seems to be important because of the addition of organic lead compounds to gasoline. However, less than 10 percent of the organic lead compounds added to gasoline are emitted in the exhaust as the organic form (Bryce-Smith and Waldron, 1974).

Most freshwater fish contain at least about 0.5 ppm of lead, with values reported as high as 16.0 ppm in green sunfish (*Lepomis cyanellus*) (Illinois, 1972). No spectacular lead values or events involving lead in fish have been reported. However, freshwater fish might possibly be of use as biological monitors. Saltwater fish contain on the average less lead than do freshwater fish; reported values range from lows around 0.1 ppm (Vinogradov, 1953; Stapleton, 1968) to highs of several ppm. Examples are Atlantic cod (*Gadus morrhua*) which contains (based on dry weight) 1-2 ppm in muscle, and 3.0 ppm in liver, and sand dab (*Pleuronectes limanda*) containing 14.0 ppm in muscle (Stenner and Nickless, 1974).

A recent detailed study of lead contamination of tuna (albacore - *Thunnus alalunga*) has revealed some interesting facts. Patterson (1977) and his coworkers sampled and analyzed albacore under ultra-clean laboratory conditions to avoid secondary contamination. Using the highly sensitive analytical method of stable isotopic dilution they were able to show that albacore muscle contained only 0.0003 ppm of lead, while commercially processed and canned tuna, analyzed by the same group, contained 0.007 ppm of lead for samples taken from non-soldered cans and 1.6 ppm for samples taken from soldered cans. Similarly, whole anchovies removed from albacore stomach contained 0.021 ppm of lead whereas commercially processed anchovies taken from soldered cans contained 4.2 ppm. The author uses these data to demonstrate the extent of lead contamination of food during processing and to emphasize that present lead levels in the biosphere are already several orders of magnitude higher than they were before man-caused lead pollution started.

The lead content of birds covers a wide range. Of specific concern is waterfowl which ingest dispersed lead shot. It has been estimated (Stickel, 1969) that over 1 million geese, ducks and swans die per year due to lead shot poisoning. Lead values reported for waterfowl liver tissue range from 0.4 to 1.5 ppm (wet weight) in ducks (Bagley and Locke, 1967) to 45 ppm for mallards in Sweden (Erne and Borg, 1969). Similarly, lead values based on wet weight vary for muscle from 2.2 to 55.8 ppm in mallards (Benson et al., 1974), for bone from 2 to 19 ppm in American scoter (Bagley and Locke, 1967), and for kidney from 4 to 99 ppm for mallards and up to 350 ppm for swans (Erne and Borg, 1969).

The lead values found in the tissues of terrestrial birds are, in general, lower although some high values have been reported. In ring-necked pheasant, lead in liver and kidney ranged from 0.09 to 0.84 ppm and 0.11 to 0.27 ppm (wet weight), respectively, while for a dying pheasant the values amounted to 169 ppm for the liver and 42 ppm for the muscle (Natl. Acad. Sci., 1972). Lead values determined for tissues of birds of prey do not differ much from their prey. Liver values for horned owl, bald eagle and osprey ranged from 0.6 to 2.8 ppm (wet) (Benson et al., 1974; Bagley and Locke, 1967) and 17.4 ppm (wet) for prairie falcon; bone values were from 1.5 ppm for osprey up to 36.0 ppm (wet) for falcon (Benson et al., 1974; Bagley and Locke, 1967).

Lead levels in mammals generally reflect their dietary habits and, naturally, the degree of environmental contamination. The influence of the traffic patterns from nearby highways on the lead content of various rodents was determined in a study conducted in Illinois (Illinois, 1974). It was found that for short-tailed shrews, least shrews, and prairie voles the relative lead values were approximately 4:2:1 for heavy, medium and low traffic areas. The values for heavy traffic areas ranged from 8.2 ppm (dry weight) for voles to 15.2 ppm (dry weight) for short-tailed shrews. In mice, white-footed mice, and deer mice, the influence of the traffic conditions was less pronounced.

Lead poisoning is one of the most frequently reported causes of poisoning in farm animals. Of major concern is the acute form of lead poisoning. It has been discussed earlier that translocation of lead from soil to plants does take place, and there is the possibility of lead entering the feed from soil. However, the bulk of the lead contamination of feed seems to result from deposition of particulates on plant surfaces.

Ruminants are more often affected by lead poisoning (Ammerman et al., 1973) than horses, poultry and swine (Blood and Henderson, 1968). The susceptibility of individual animals depends on the type of lead compound (mainly if inorganic or organic), kind of animal, age, health, etc. As little as 6 mg lead/kg of body weight given daily over 60 days has been fatal for cattle (Hammond and Aronson, 1964). Approximately 0.2 to 0.4 g/kg body weight of lead on any one day, ingested as acetate, basic carbonate or oxide caused death in calves up to 4 months of age (Allcroft, 1951). Older cattle can tolerate twice this dose (Buck, 1970). Marshall et al. (1963), in their studies on lead transfer to bovine milk, fed lactating cows up to 13 mg of lead/kg body weight for 126 days, apparently without serious effects. Adult

sheep survived single doses of up to 0.6 g/kg while abortions and death occurred at these levels with pregnant ewes (Allcroft and Blaxter, 1950). Ewes receiving up to 4.5 mg lead/kg as finely divided metallic lead for 27 weeks including 22 weeks of gestation, showed no signs of clinical lead poisoning (Carson et al., 1973). Horses grazing near lead smelters may also be subject to lead poisoning because, in addition to the aerial plant parts, they ingest the roots and the adhering lead-contaminated soil particles, both often containing much more lead than the plant tops (Schmitt et al., 1971).

Lead distribution in mammalian tissue depends on the chemical form of the lead ingested, the animal species and the administrative route (Natl. Acad. Sci.. 1972; Blaxter, 1950a, 1950b). Lead absorbed from the gut goes mainly to bone and kidney while injected lead goes to bone marrow, spleen and liver. Apparently orally ingested lead is primarily deposited in the skeleton until the threshold value is attained and then it is deposited in other tissues, particularly the kidneys (Cantarow and Trumper, 1944; Natl. Acad. Sci. 1972). Schroeder and Balassa (1961a) reported lead content in cow muscles (wet weight) of 0.20 ppm, with 0.67 and 0.51 ppm present in liver and kidney, respectively. Allcroft (1950) found up to 126.0 and 297.0 ppm of lead in the kidney and liver, respectively, from cows that had ingested lead-containing materials. In sheep muscle 0.15 ppm (wet weight) of lead was found, and values reported for pigs were 0.16 ppm for muscle, 0.26-0.82 ppm for liver and 0.26-0.98 ppm for kidney (Schroeder and Balassa, 1961a). Horses that ate contaminated grass near a smelter had lead concentrations (controls in parentheses) in kidney of 40.4 (3.1) ppm, liver 12.0 (9.0) ppm and lung 1.6 (1.3) ppm (Goodman and Roberts, 1971). In experimental studies it was demonstrated that lead in guinea pigs crossed the placental barrier. The maternal femur of guinea pigs fed a diet containing 2,000 ppm of lead contained 626 ppm of lead while the fetal femur contained 5700 ppm (Illinois, 1974).

The determination of lead in hair and teeth has received special attention in recent years as a possible screening method for lead accumulation in animals and humans. Kopito and Shwachman (1975) investigated the accumulation of lead in human scalp hair in male and female children and adults from various locations in several countries. They concluded that the most significant variables which influenced the concentration of lead in hair were ingestion of lead-containing substances, exposure to lead of environmental origin, place of residence, location of sample along the hair shaft relative to its distance from the scalp, and age. Hair clippings have also been used to test 300 animals for lead (Bazell, 1971). A major problem in the use of hair samples for determining lead exposure is the tendency of hair to accumulate surface lead from the atmosphere. This and other uncertainties mentioned earlier require that more work be done before hair can be used as a reliable screening and monitoring tool for lead. Altshuler et al. (1962) analyzed deciduous teeth from children who died from lead poisoning, teeth from lead-poisoned but surviving children, and shed teeth from control children. The mean levels found were 160 ppm for fatal cases, 116 ppm for poisoned but surviving children, and 15 ppm for the control children. Needleman et al. (1972) analyzed deciduous teeth of children who lived in the "Lead Belt" of urban Philadelphia and from Philadelphia suburbs, and found that the lead content of the teeth of the former was 51.1 ± 109.0 ppm and 11.1 ± 14.8 ppm

for the suburban controls. The authors claim that deciduous teeth can be used to identify past exposure, as a possible means to measure hidden deficits caused by lead ingestion. Wilkinson and Palmer (1975) found in a study that the amount of lead contained in human teeth increased at a fairly uniform rate with age up to about 50 years at which time a sharp increase was noted. Human teeth collected from people living in a rural area in Delaware showed a lower accumulation of lead than teeth collected from people living in the inner city area of Baltimore. The above results appear encouraging but more research is required before teeth can be used as a reliable monitoring means for lead exposure.

ARSENIC

Arsenic is ubiquitous in the environment. The most important man-related sources of arsenic pollution are smelters which emit arsenic compounds from their stacks, and the use of arsenic compounds as pesticides and herbicides. The latter practice has been largely discontinued but very high arsenic levels in the soil have accumulated in areas of earlier high applications of arsenic pesticides (Woolson et al., 1971).

Plants can take up arsenic from soil but there appears to be little danger of poisoning to the animals which consume these plants; however, animals grazing on plants contaminated externally by arsenic trioxide have died (Haywood, 1907). Also, injuries to humans living in communities exposed to industrial arsenic contamination have been reported (Oyanguren and Perez, 1966; Birmingham et al., 1965). Perhaps the most important aspect of concern with arsenic in the environment is the potential carcinogenicity of arsenical compounds to humans.

Freshwater concentrations of arsenic are usually in the ppb range, but values in the ppm range have been reported (EPA, 1976). Arsenic concentrations reported for freshwater fish are usually below 1 ppm (wet weight) with 0.09 ppm reported for freshwater drum fish (Pillay et al., 1974), 0.52 ppm for bluegills (Gilderhus, 1966), 0.069-0.149 ppm for trout (Pratt et al., 1972), 0.055-0.51 ppm for carp (Ellis et al., 1941; Pratt et al., 1972) and 0.8 ppm for pike (Chapmann, 1926). Values as high as 2.75 ppm have been reported for small-mouthed buffalo fish and up to 77.31 ppm for the liver oil of the large-mouth black bass (Ellis et al., 1941).

Seawater contains several ppm of arsenic (EPA, 1976). The arsenic content of marine fish is generally higher than that of freshwater fish. Arsenic values (based on wet weight) for anchovies ranged from 7.1 to 10.7 ppm (Lunde, 1973), for tuna from 0.71 to 4.6 ppm (Cardiff, 1937; Orvini et al., 1974); sole contained 5.2 ppm (Chapman, 1926) and herring fillet 3.8 ppm (Lunde, 1970). Lunde (1970) analyzed extracts of fish muscle and found that the aqueous fraction contained most of the arsenic. His reported values ranged from 0.9 ppm (based on dry weight) of arsenic for whale extract to 37 ppm for cod liver extract. A standard reference material which is available from the International Atomic Energy Agency ("Fish Solubles A-6, 1974") is certified for 14.5 ppm of arsenic.

Some marine organisms tend to accumulate arsenic. Schrenk and Schreibeis (1958) reported arsenic values of 3-10 ppm for oysters, 70 ppm for lobster, up to 120 ppm for mussels, and 170 ppm for prawns. The same authors note that the arsenic content of urine in humans is generally higher following the consumption of seafood.

Relatively few data are available on the arsenic content of wild birds. Andren et al. (1973) reported arsenic levels, based on dry weight, of 0.05 ppm for owl, 0.1 ppm for crow, 0.2 ppm for sparrow, and 0.4 ppm for hawk. Domestic birds, mainly chicken, can be fed growth-promoting drugs which contain arsenic, so that maximum permissible levels of arsenic have been established for poultry which are 0.5 mg/kg for fresh, uncooked muscle, and 2.0 mg/kg for fresh, uncooked by-products (Woolson, 1975). Obviously, the levels found in usable poultry tissue must be lower than the maximum permissible amount. However, since there is an increasing interest in the use of dried poultry excreta as cattle feed supplement, the levels of the arsenic contents of poultry wastes as well as the effect of arsenic on ruminants are of special interest. The FDA has not granted approval of the use of arsenicals in the feed for ruminants.

Andren et al. (1973) determined arsenic levels, based on dry tissue weight, for wild mammals. They reported 0.2 ppm of arsenic for opossum, 0.8 ppm for squirrel and fox, and 1.0 ppm for mice. Arsenic values reported for domestic mammal tissue include 0.063 ppm for beef liver (Orvini et al., 1974), 0.22-0.32 ppm for swine muscle (Barela and Pezzeri, 1966; Pezzeri, 1970), and 0.52 for calf muscle (Pezzeri, 1970). Swine can be fed growth-promoting arsenic-containing drugs; studies have shown that the bulk of the ingested arsenicals was rapidly eliminated, once administration of the drugs was stopped (Woolson, 1975).

In a number of studies, the distribution of arsenic in animals has been investigated using radioisotopes of arsenic. It was found that in most animals arsenic was present in all tissues, with the highest accumulation in the muscles (EPA, 1976). Only in rats was arsenic concentrated in the red blood cells, which makes rats rather undesirable as a model for studies with arsenic (Hunter et al., 1942; Ducoff et al., 1948; Lanz et al., 1950).

Arsenic concentrations in washed hair ranged from 1 to 5.5 ppm (Perkons and Jervis, 1966; Dubois et al., 1965). The question of whether the arsenic content of hair is indicative of previous arsenic ingestion received widespread publicity when the hair of Napoleon was analyzed via neutron activation and was found to contain arsenic. It was determined that there was a good correlation between the distribution of arsenic along the length of the hair shaft and the ups and downs of Napoleon's health (Forshufvud et al., 1961; Smith et al., 1962; Forshufvud et al., 1964). Arsenic is transferred to animal and human hair, but it must be remembered that external contamination of hair by atmospheric dust can readily occur. Dubois et al. (1965) found arsenic values in hair of up to 243 ppm, but after washing the hair in detergent, all values were reduced to 3.0 ± 1.0 ppm. Contrary to this it was also reported that arsenic in the environment reacts with the keratin of the hair and cannot be removed by repeated washings (Lerner, 1954). When hair from cases

of arsenic poisoning was examined, a concentration of 3.0 to 26.0 ppm was found shortly after arsenic exposure (Lander et al., 1965). The concentrations near the hair tips were often similar to those near the scalp, a fact which the authors attributed to arsenic-deposition from sweat. It is obvious that a number of problems need to be addressed before hair can be used to monitor the previous arsenic exposure of individuals.

CADMIUM

The cadmium accumulation and distribution in animal tissue have been investigated to a lesser extent than those of mercury, lead and arsenic. Cadmium is an accumulative poison, and its long-term biological effects are not well understood (Hiatt and Huff, 1975). However, cadmium is suspected of being carcinogenic to man as well as teratogenic and mutagenic, as has been stated earlier. This fact combined with the fairly wide distribution of cadmium makes it necessary to monitor its concentration levels in the environment.

In nature, cadmium is usually associated with zinc. Zinc and cadmium are known to be antagonistic to each other in humans (Bunn and Matrone, 1966; Underwood, 1971); however, the protective action of zinc does not reduce absorption of cadmium and its transfer to tissues. Some of the toxic effects of cadmium can be reduced by selenium, cobalt, and certain sulfur compounds (Flick et al., 1971; Friberg et al., 1971).

The cadmium levels reported for freshwater fish tissue are generally low. Values ranged from less than 0.01 ppm (wet weight) for carp and white bass to 0.142 ppm for goldfish collected from the Hudson River (Lovett et al., 1972) and 0.14 ± 0.06 ppm for trout-perch (Lucas et al., 1970). Values as high as 23 ppm were reported for brook trout (Lovett et al., 1972). Cadmium levels in freshwater fish livers are somewhat higher, ranging for lake trout liver from a low of 0.06 ± 0.02 ppm to a high of 3.0 ppm (Lucas et al., 1970). A study using bluegill showed that exposure to varying cadmium concentrations resulted in corresponding similar cadmium variations in the fish tissue (mg Cd/l vs ppm Cd in tissue): 0.008 vs 0.03, 0.08 vs 0.1, and 0.85 vs 1.1 (Cearley and Coleman, 1974). A value of 1300 ppm* (dry weight) of cadmium was reported for channel catfish exposed to sub-lethal amounts of cadmium (Mount and Stephan, 1967). Saltwater fish contain levels of cadmium similar to freshwater fish. Herring, sea trout and haddock muscle contained 0.06 ppm, 0.01 to 0.015 ppm, and 0.003 to 0.014 ppm (wet weight), respectively (Havre et al., 1973), whereas sand eel, swordfish and lanternfish contained, on a dry weight basis, 0.4 ppm, 0.9 ppm, and 1.6 ppm, respectively (Stevens and Brown, 1974; Gibbs et al., 1974). Again, cadmium accumulates in the fish liver: angler and whiting contained 0.023 ppm and 0.003 to 0.032 ppm of cadmium in the muscle, but 2.5 ppm and 0.17 ppm, respectively, in the liver (Havre et al., 1973).

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* It has been cautioned (Friberg et al., 1971) that certain salts, especially sodium chloride, can interfere with the cadmium determination via atomic absorption spectrophotometry.

Few values have been reported for the cadmium content of bird tissue. Martin and Nickerson (1973) analyzed starlings and found that in 46 areas of the United States the cadmium concentrations in starling muscle were below 0.1 ppm (wet weight) with most cases even below 0.005 ppm. Only in certain city areas did the cadmium level go as high as 0.24 ppm. Liver and kidney concentrations are generally considerably higher than corresponding muscle values. Ruffed grouse liver contained, on a wet weight basis, 0.88 to 2.04 ppm of cadmium (Schroeder et al., 1967; Schroeder and Balassa, 1961b), and pheasant, starling and robin liver contained 0.9 ppm, 0.57 ppm, and 0.55 ppm, respectively (Schroeder and Balassa, 1961b). Starling and robin kidneys contained 1.0 ppm and 2.03 ppm, respectively (Schroeder and Balassa, 1961b).

Cadmium absorption from the GI tract in mammals is low. Rats, mice and monkeys absorb approximately 2 to 3 percent of ingested cadmium while humans seem to absorb nearly 6 percent (Friberg et al., 1971; Friberg and Vostal, 1972). In experiments with goats it was found that the percentage of dietary radioactive cadmium which was absorbed and retained was the same even when the dose was increased about 400-fold (Miller et al., 1969). However, many of the cadmium absorption data reported in the literature are derived from the difference between intake and fecal excretion. Obviously when this difference is small, there is room for large errors (Miller, 1975).

The quantity of dietary cadmium which is toxic to mammals depends on such variables as animal species, dose, and method of administration (Ammerman et al., 1973; Friberg et al., 1971; Underwood, 1971). Four calves which received 2,560 ppm of dietary cadmium died after 2, 3, 5 and 8 weeks (Powell et al., 1964). Other calves survived 640 ppm of cadmium in the feed. In rats, 500 ppm of cadmium was lethal (Wilson et al., 1941). The highest cadmium concentrations are usually found in the kidney, followed by the liver. When radioactive cadmium was administered to goats, 50 percent of the total body burden was found in the liver and 23 percent in the kidneys (Miller et al., 1969). Under similar conditions, lactating cows accumulated 32 percent in the liver and 10 percent in the kidneys (Neathery et al., 1974).

The administration route has a profound influence on the relative distribution in the tissue. This was demonstrated by Miller and his associates (1968) who administered radioactive cadmium to young goats and found that two weeks after oral administration, the cadmium concentration in the muscle was 7.4 percent of that in the liver while 2 weeks after an intravenous dose, the muscle tissue contained only 0.4 percent of that in the liver. In general, dietary cadmium seems to preferentially accumulate in the kidney, and intravenously administered cadmium concentrates in the liver.

Cadmium levels in blood and muscle tissue are low, independent of the mode of administration (Neathery and Miller, 1975). Thus, muscle, the most important tissue for human consumption, is well protected from ingested cadmium.

Some values for cadmium levels in the liver and kidney of wild land mammals have been published. Schroeder et al. (1967) found 0.36 ppm (wet weight) in coyote liver and 0.73 ppm (wet weight) in red squirrel liver, and Schroeder and Balassa (1961b) reported 0.3 ppm for rabbit liver. Published kidney values are generally higher. Schroeder et al. (1967) reported 2.07 ppm

of cadmium for deer kidney, and Schroeder and Balassa (1961b) found 3.62 ppm in gray squirrel kidney, 7.97-17.35 ppm in red squirrel kidney and 3.58 ppm in rabbit kidney.

Cadmium levels in the liver and kidneys of domestic mammals are similar. Schroeder et al. (1967) found 0.28 ppm of cadmium in cow liver and 0.52 ppm in cow kidney. Schroeder and Balassa (1961b) reported 0 ppm which means below the detection limit for pig liver, 0.15-0.6 ppm for pig kidney, and 0.14 for sheep kidney, Goodman and Roberts (1971) reported 1.6 ppm for horse liver and 35.0 ppm for horse kidney, and Doyle et al. (1974) found up to 769 ppm in sheep kidney and 276 ppm in sheep liver after feeding sheep high cadmium doses.

Small amounts of cadmium are found in hair. Hammer et al. (1971) determined cadmium levels in the hair of fourth-grade boys in several cities and concluded that mean hair cadmium levels reflect community exposure. However, they cautioned that it is not fully known how well hair reflects the body burden of the metal. The arithmetic means of cadmium content of the boys' hair ranged from 0.8 to 2.1 ppm, depending on the city. Petering et al. (1973) concluded from experimental studies that the cadmium content of hair of the general population not exposed occupationally ranges between 0.5 and 2.5 ppm, regardless of sex and age above 2 years. Eads and Lambdin (1973) analyzed human hair samples collected in an industrial city and reported mean cadmium concentrations of 2.2 and 1.0 ppm for hair of males and females, respectively. The latter studies support the hypothesis that age influences the cadmium content of human hair.

CONSIDERATIONS FOR THE PREPARATION OF ANIMAL TISSUE SRM's

A wide range of toxic element concentrations occur in animal tissues. This is illustrated in Figure 2 where the ranges of mercury, lead, arsenic and cadmium found in animal tissues, as discussed in this report, are presented. Included are the corresponding analytical values for these elements as certified for the Bovine Liver SRM available from the NBS.

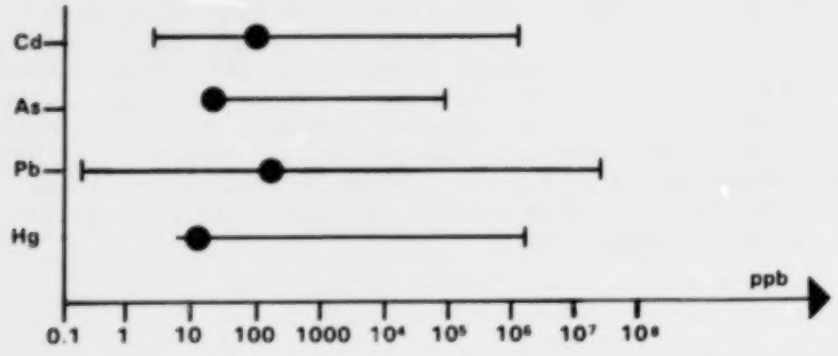

Figure 2. Ranges of mercury, lead, arsenic, and cadmium levels as reported in the literature for animal tissues.

It is obvious that a need exists to prepare a series of SRM's that vary in the concentrations of the toxic elements under consideration to more closely cover the concentration ranges encountered in the samples. The low value reported for lead in albacore was determined under very special conditions that cannot usually be achieved in analytical laboratories. Therefore, no need exists at present to prepare SRM's that cover this extremely low lead level. However, it should be understood that relatively high levels of lead and other toxic elements in SRM's do not imply that these levels are environmentally acceptable. Rather, these levels are essential for the usefulness of SRM's in conjunction with contaminated samples that might even contain toxic levels of pollutants.

It has been discussed earlier that different pollutants administered to animals may accumulate in different target tissues. This makes it desirable

to collect and process a variety of animal tissues to be used as SRM's, such as liver, kidney, muscle, and bone. Liver and kidney concentrate a wide variety of pollutants, bone accumulates specific pollutants such as lead, and muscle is the most important tissue for human consumption. It is desirable to also prepare SRM's from other animal tissues which can easily be collected in the environment for screening and monitoring purposes. Such tissues include blood, hair, and possibly brain, teeth, and hoof.

In order to get a relatively fast accumulation of toxicants in certain tissues such as bone or teeth, young animals which are still in the active growth phase should be used, with the toxicants administered over prolonged periods of time. It has been discussed earlier that the metabolic fate and the tissue distribution of toxicants may vary with the mode of dose application. Since in the environment most pollutants enter the animal body via the digestive tract, it is reasonable to apply the toxicants orally rather than intravenously. Animals used for the in vivo incorporation of toxicants should have at the least average resistance to the toxicants to be administered, they should be easy to handle and maintain, and they should not be expensive.

These requirements narrow the selection to domestic or experimental mammals. Experimental animals commonly used in laboratories, such as mice, rats, guinea pigs and rabbits, are small in size and a large number would be required to produce an adequate amount of processible tissue. Furthermore, the dosing of the animals as well as cleaning the small organs following sacrifice are cumbersome and labor-intensive and thus costly. The attractive feature for the use of animals such as bovine or horse is their large size. Dose administration would be relatively simple, sufficient tissue could be recovered from just one or a few animals at the most, and tissue preparation would pose no problem. However, because of the animals' size, facilities would be required which might not be readily available. In addition to this, the small number of animals needed to obtain the required amount of tissue may become a disadvantage should an illness or injury occur among the animals during the experiment. A compromise of sizes might be the most suitable and cost-effective solution. Several intermediate sizes of domestic animals are available such as sheep, goats, and large and miniature pigs. Pigs are poor hair-growers and can pose an odor and handling problem. Goats and sheep seem equally suitable with goats having a slight advantage, because of their better resistance to disease, and because they grow hair instead of wool.

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APPENDIX A:

MERCURY CONTENT OF SELECTED PLANT AND ANIMAL TISSUES

TABLE 1. MERCURY CONTENT OF ALGAE

Species[a]	ppm, D/W[b]		Reference
Ascophyllum nodosum	0.319	D	Jones et al. (1972)
Caldophora rupestris	0.826	D	"
Ceramium rubrum	3.031	D	"
Enteromorpha compressa	1.007	D	"
Fucus serratus	1.153	D	"
Fucus vesiculosus	0.083-0.206	D	"
Laminaria digitata	0.794	D	"
Polysiphonia lanosa	0.612	D	"
Porphyra umbilicalis	2.353	D	"
Ascophyllum nodosum	0.05- 1.2	D	Stenner and Nickless (1974)
Fucus vesiculosus	0.018-0.023	W	Stock and Cucuel (1934)
Ulva pertusa	5.3- 14.00	D	Matida and Kumada (1969)
Phytoplankton, Great Lakes	0.44	W	Copeland (1972)
" " "	2.2	D	"

(a) As named by authors

(b) Based on dry (D) or wet (W) weight of the plant material

TABLE 2. MERCURY CONTENT OF FUNGI

Species[a]	ppm, Dry Weight	Reference
Agaricus arvensis	0.4 - 1.1	Stegnar et al. (1973)
Boletus edulis	2.2 - 2.4	"
Clavaria sp.	6.6 -16.4	"
Hypholoma sp.	15.5 -20.2	"
Kuehneromyces mutabilis	0.35- 0.92	"
Lactarius sp.	80.5	"
Lycoperdon perlatum	32.2 -74.0	"
Polporus sp.	0.4	"
Russula sp.	23.9 -36.0	"
Scleroderma vulgare	0.01- 0.02	"
Boletus subtomentosus	0.24- 4.0	Rantes (1975)[b]
Collybia butyracea	1.90-62.5	"
Collybia confluens	3.39-73.6	"
Cortinarius odorifer	1.90-19.5	"
Lactarius delitiosus	0.68- 7.9	"
Lactarius scrobiculatus	0.14- 3.0	"
Lepista nuda	1.50- 5.2	"
Lycoperdon gemmatum	5.54-64.3	"
Lycoperdon piriformi	2.60- 5.9	"
Mycena pura	4.20-44.8	"
Ramaria invalia	0.14- 2.2	"
Rusulla integra	0.04- 1.8	"
Tricholoma scalpturatum	5.6 -86.5	"

(a) As named by authors

(b) High values derived from samples collected within 1 km of chloralkali plant

TABLE 3. MERCURY CONTENT OF MOSSES

Species[(a)]	ppm, Dry Weight	Reference
Fontinalis sp.	3.70	Wallace et al. (1971)
Eurhynchium hians	0.012-0.080	Huckabee (1973)
Brachythecium rivulare	0.012-0.080	"
Sharpiella striatella	0.012-0.080	"
Dicranum sp.	0.118	Huckabee and Blaylock (1973)
Polytrichum	0.092	" "

(a) As named by authors

TABLE 4. MERCURY CONTENT OF AQUATIC VASCULAR PLANTS

Species[a]	Plant Part	ppm, D/W[b]		Reference
Alligator weeds	Whole	149	D (max.)	Wolverton and McDonald (1975a)
Elodea densa	Whole 10 ppm CH_3Hg^+ in H_2O	1000	W	Mortimer and Kudo (1975)
"	Whole 10 ppm Hg^{2+} in H_2O	400	W	" "
Scirpus cyperinus (in .04 µg Hg/l H_2O)	Submerged stem	1.02	W	Eriksson and Mortimer (1975)
	Root	0.23	W	" "
Sagittaria latifolia	Leaf	0.3	W	" "
"	Stem	0.49	W	" "
"	Root	0.04	W	" "
Water hyacinth	Whole	151	D	Wolverton and McDonald (1975a)
"	Leaves	∿15	D	Wolverton and McDonald (1976)
Ceratophyllum demersum		$\leq$ 6.95	D	Fang (1973)
Elodea canadensis		$\leq$ 9.35	D	"
Water lily		0.52	D	"

(a) As named by authors

(b) Based on dry (D) or wet (W) weight of the plant material

TABLE 5. MERCURY CONTENT OF TREE LEAVES

Species[(a)]	ppm, Dry Weight	Reference
Sugar maple	0.81	Smith (1972)
Norway spruce	0.22	"
Australian pine	0.17	"
London plane	0.71	"
Pin oak	0.76	"
Basswood	1.10	"
Red cedar	<0.5	Shaklette (1970)
Black spruce	1.0-1.5	"
Rosa sp.	1.3-808.0	Stahl (1969)
Orchard leaves	0.155	NBS (1975)

(a) As named by authors

TABLE 6. MERCURY CONTENT OF FRUIT

Species[a]	ppm, Wet Weight	Reference
Apple, red	0.007-0.025	Gerdes et al. (1974)
Apple, yellow	0.083-0.092	"
Banana	0.032-0.147	"
Cherry	0.004-0.014	"
Grape	0.028-0.034	"
Lemon	0.087-0.135	"
Lime	0.075-0.158	"
Melon	0.006-0.013	"
Nectarine	0.094-0.1	"
Orange	0.074-0.102	"
Peach	0.053-0.057	"
Pear	0 -0.092	"
Plum	0.047-0.282	"
Strawberry	0.043-0.053	"

(a) As named by authors

TABLE 7. MERCURY CONTENT OF VEGETABLE PLANTS

Species[a]	ppm, Wet Weight	Reference
String beans	0.046-0.057	Gerdes et al. (1974)
Broccoli (head)	0.024-0.027	"
Cabbage (head)	0.027-0.123	"
Carrot (root)	0.004-0.006	"
Cauliflower (head)	0.020-0.046	"
Celery (stalks)	0.007-0.023	"
Cucumber (fruit)	0.002-0.019	"
Eggplant (fruit)	0.045-0.048	"
Lettuce (leaves)	0.019-0.021	"
Okra (pod)	0.057-0.097	"
Onion, white (tuber)	0.033-0.049	"
Pepper, green	0.001-0.1	"
Potato (tuber)	0.026-0.042	"
Radish (root)	0.001-0.007	"
Squash (fruit)	0.001-0.005	"
Sweet potato (tuber)	0.019-0.036	"
Tomato (fruit)	0.02 -0.036	"

(a) As named by authors

TABLE 8. MERCURY CONTENT OF GRAIN CROPS

Species[a]	ppm, D/W[b]		Reference
Barley	<0.02	D	Smart (1968)
"	0.03	D	D'Itri (1972)
Corn	0.006-0.033	W	Gerdes et al. (1974)
Rice - Japan	0.23 -1.0	D	Smart (1968)
" Texas	0.08 -0.092	W	Gerdes et al. (1974)
Wheat	0.008-0.012	D	Smart (1968)
"	0.005-0.040	D	Saha (1972)

(a) As named by authors

(b) Based on dry (D) or wet (W) weight of the plant material

TABLE 9. MERCURY CONTENT OF FRESHWATER FISH

Species[a]	Location	Tissue	ppm[b]	Reference
Rock bass *Ambloplites rupestris*	Michigan	muscle	(1.14-10.90) 6.22	Fimreite and Reynolds (1973)
American eel *Anguilla rostrata*	Chesapeake Bay	muscle	(0.02-0.12)0.06	Bender et al. (1972)
White fish *Coregonus clupeaformis*	Lake Huron	edibles	0.05-0.15	Rottschafer et al. (1971)
Northern pike *Esox lucius*	Ontario	muscle	1.61-27.8	Fimreite and Reynolds (1973)
	E. Canada	"	1.40	Fimreite et al. (1971)
Bluegill *Lepomis macrochirus*	Michigan	edibles	0.40	Rottschafer et al. (1971)
Smallmouth bass *Micropterus dolomieui*	New York	whole fish	0.55	Bache et al. (1971)
Largemouth black bass *Micropterus salmoides*	Utah	muscle	(0.17-7.3)1.94	Smith (1973)
Yellow perch *Perca flavescens*	Utah	"	(0.13-0.43)0.29	"
Brown trout *Salmo fario*	near cinnabar refinery	muscle	0.08-9.6	Byrne et al. (1971)
Rainbow trout *Salmo gairdnerii*	experiment exposed to Hg phosphate	blood	22.8	Rucker and Amend (1969)
"	"	kidney	17.3	"
"	"	liver	16.7	"
"	"	brain	10.1	"

(Continued)

TABLE 9. MERCURY CONTENT OF FRESHWATER FISH (Continued)

Species[a]	Location	Tissue	ppm[b]	Reference
Lake trout *Salvelinus namaycush*	E. Canada	muscle	1.07-10.5(5.78)	Fimreite et al. (1971)
	New York	whole fish	0.14-0.16	Bache et al. (1971)
Walleye pike *Stizostedion vitreum*	Ontario(normal)	muscle	0.24- 1.12	Fimreite and Reynolds (1973)
	Ontario(polluted)	"	0.28-19.6	" "

(a) As named by authors

(b) Based on wet tissue weight

TABLE 10. MERCURY CONTENT OF MARINE FISH

Species[a]	Location	Tissue	ppm[b]	Reference
American shad *Alosa sapidissima*	Virginia	flesh	0.10	Boyle (1970)
Atlantic herring *Clupea harengus*	Atlantic	muscle	0.07±0.01	Simpson et al. (1974)
Haddock *Gadus aeglefinus*	"	muscle	0.04±0.01	"
Common cod *Gadus morrhua*	"	muscle	0.09±0.04	"
Halibut *Hippoglossus hippoglossus*	Sweden Atlantic	muscle "	0.026-0.036 0.14±0.03	Westöö (1967) Simpson et al. (1974)
Pacific blue marlin *Makaira ampla*	Hawaii	muscle liver	(0.35-14.0)4.78 (0.39-36.0)7.57	Rivers et al. (1972)
White perch *Morone americana*	Chesapeake Bay	muscle	(0.02-2.0)0.1	Bender et al. (1972)
Striped bass *Morone saxatilis*	"	"	(0.08-0.22)0.13	"
Flounder *Pleuronectes flesus*	Atlantic	muscle	0.08±0.03	Simpson et al. (1974)
Sardine *Sardinia pilchardus*	"	"	0.02±0.01	"
Atlantic mackerel *Scomber scombrus*	"	"	0.12±0.11	"

(Continued)

TABLE 10. MERCURY CONTENT OF MARINE FISH (Continued)

Species[a]	Location	Tissue	ppm[b]	Reference
Albacore tuna *Thunnus albacora*	(domestic)	canned tuna	0.25±0.1	Simpson et al. (1974)
Swordfish *Xiphias gladius*	California	muscle	0.23-1.27	Miller et al. (1972)

(a) As named by authors

(b) Based on wet tissue weight

TABLE 11. MERCURY CONTENT OF BIRDS

Species[a]	Location	Tissue	ppm[b]	Reference
(A) Water birds:				
Spotted sand piper	United States	carcass	0.55	Dustman et al.
Actitis macularia	"	liver	2.8	(1972)
Mallard	"	liver	0.23- 4.8	"
Anas platyrhynchos	"	kidney	0.1 - 3.5	"
"	"	muscle	0.1 - 1.15	"
Great blue heron	"	liver	14.6 -175.0	"
Ardea herodius	"	carcass	5.3 - 23.0	"
Common egret	"	liver	6.3	"
Casmerodius albus	"	carcass	0.74	"
Common eider	Finland	liver	12.9	Henriksson et al.
Somateria mollissima	"	kidney	1.6	(1966)
"	"	muscle	3.9	"
(B) Birds of prey				
Goshawk	Sweden	liver	6.0 - 53.0	Borg et al. (1966)
Accipiter gentilis				
Buzzard	Norway	kidney	0.3	Holt (1969)
Buteo buteo				
Bald eagle	United States	carcass	59.0	Mulhern et al.
Haliaeetus leucocephalus	"	brain	130.0	(1970)

(Continued)

TABLE 11. MERCURY CONTENT OF BIRDS (Continued)

Species[a]	Location	Tissue	ppm[b]	Reference
(C) Terrestrial birds				
Hooded crow	Sweden	liver	35.0	Westermark (1967)
Corvus corone	"	kidney	28.0	"
cornix	"	muscle	18.0	"
"	"	brain	12.5	"
Gambels quail	Arizona	liver	over 0.5	Montague (1971)
Lophortyx gambelii	"	muscle	below 0.2	"
Black grouse	Norway	kidney	0.68	Holt (1969)
Lyrurus tetrix				
Ring-necked pheasant	California	muscle	1.6 - 4.7	Benson et al. (1971)
Phasianus colchicus	Colorado	"	0.04 - 0.6	Montague (1971)
"	Idaho	"	0 -15	"
"	Idaho	"	0.16	Benson et al. (1971)
"	Indiana	"	0.058	Montague (1971)
"	Oregon	"	to 0.5	"
"	Utah	"	0.01 - 2.08	Smith et al. (1974)
"	Canada	"	0.006- 0.46	Jervis (1970)
"	Denmark	"	0.01	Berg et al. (1966)
"	Finland	"	0 -13.4	Karppanen et al. (1970)
Starling	Sweden	liver	2.2 -21.0	Westermark (1967)
Sturnus vulgaris	"	kidney	2.4 -24.3	"
"	"	muscle	0.6 - 5.7	"
"	"	brain	1.0 - 9.2	"

(a) As named by authors

(b) Based on wet tissue weight

TABLE 12. MERCURY CONTENT OF MAMMALS

Species[a]	Location	Tissue	ppm[b]	Reference
Northern fur seal	Washington	liver	7.1 -172.0	Anas (1974)
Callorhinus ursinus	"	kidney	0.6 - 1.6	"
"	"	muscle	0.2 - 0.4	"
Harbor seal	Calif.	liver	81.0 -700.0	"
"	Oregon.	"	0.3 - 68.0	"
"	Wash.	"	1.3 - 60.0	"
"	Pribilof Island	"	0.6 - 8.9	"
"	Nova Scotia	fur	1.8	Freeman and
"	"	liver	0.99	Horne (1974)
"	"	kidney	0.67	"
"	"	muscle	0.55	"
Coyote	Wyoming	hair	to 0.6	Huckabee et al.
Canis latrans				(1973)
Red-backed mice	"	hair	to 0.6	"
Clethrionomys gapperi				
Amer. mink (normal diet)	United States	liver	0.28±0.06	Aulerich et al.
Mustela vison	"	kidney	0.68±0.14	(1974)
"	"	muscle	0.05±0.03	"
"	"	spleen	0.24±0.22	"
"	"	brain	0.22±0.14	"
Black bear	Idaho	hair	0.11- 0.275	Benson et al.
Ursus americanus	"	muscle	0.04- 0.171	(1974)
"	"	fat	0.05- 0.12	"

(Continued)

TABLE 12. MERCURY CONTENT OF MAMMALS (Continued)

Species[a]	Location	Tissue	ppm[b]	Reference
Reindeer	Sweden	liver	0.004-0.27	Westöö (1969)
Rangifer tarandus	"	kidney	0.002	"
"	"	muscle	0.005-0.023	"

U.S. GOVERNMENT PRINTING OFFICE: 1978 - 786-154/1269 Region No. 9-1